Acute Toxicity of Vomitoxin (Deoxynivalenol) in Broiler Chickens[1,2]

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ABSTRACT Acute vomitoxicosis in broiler chicks was characterized by extensive ecchymotic hemorrhaging throughout the carcass, widespread deposition of urates, disturbance of the nervous system, and irritation of the upper gastrointestinal tract. The approximate oral LD_{50} dose for vomitoxin was 140 mg/kg, suggesting substantially lower toxicity than with aflatoxin or ochratoxin.
(Key words: vomitoxin, acute toxicity, hemorrhaging, chickens, urate deposition)

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INTRODUCTION

Vomitoxin is a trivial name for deoxynivalenol or 3,7,15-trihydroxy-12,13-expoxytrichothec-9-en-8-one (Ueno, 1977). It is a mycotoxin produced in corn by *Fusarium* species (Vesonder *et al.*, 1976) and, as its name suggests, it is a powerful emetic agent with minimal emetic doses in swine of .05 mg/kg intraperitoneally and .1 mg/kg orally (Forsith *et al.*, 1977). The major economic loss associated with vomitoxin appears to be that of feed refusal in swine, which has been a problem of swine husbandry in the Midwest for 50 years (Dickson *et al.*, 1930). The effects of vomitoxin in chickens have not been reported; indeed chickens do not possess the musculature needed for active emesis. Nevertheless, vomitoxin is closely related chemically to T-2 toxin, which has severe and dramatic effects in chickens (Doerr *et al.*, 1974; Wyatt *et al.*, 1975a,b). The present study was initiated to gain preliminary information for a possible diagnosis of vomitoxicosis in chickens and for the need of a more extensive evaluation of this mycotoxin, which is difficult to produce in the pure state.

MATERIALS AND METHODS

Day-old male broiler chicks (Cobb × Cobb) were housed in electrically heated batteries under continuous illumination with feed and water available *ad libitum*. Crystalline vomitoxin was produced by the method of Vesonder *et al.* (1976). Known amounts of pure vomitoxin were dissolved in distilled water and administered to the day-old chicks *via* crop intubation at the dose levels of 0, 35, 70, 140, 280, 560, and 1120 mg/kg body weight. Two birds per treatment were used. Mortality by the end of 7 days was recorded and the survivors were necropsied. The time of deaths was recorded and necropsies performed on the carcasses. Symptoms were recorded when they occurred.

RESULTS AND DISCUSSION

The mortality caused by vomitoxin is shown in Table 1. An accurate LD_{50} value cannot be estimated because of the lack of variation, but the data indicate the value is near 140 mg/kg. Immediately after administration of toxin, the birds began to gasp, became lethargic, assumed a squatting position, and dropped their wings and head from the normal upright position. This behavior was followed by a loss of balance and righting reflex. These neural disturbances resembled those seen during T-2 toxicosis (Wyatt *et al.*, 1973) except that spontaneous tremors and hysteroid seizures induced by noise were not observed with vomitoxin. As the toxicosis progressed the birds would raise their heads, shake themselves, and swallow, a behavior pattern associated with irritation of the upper gastrointestinal tract (J. R. Harris, personal communication). This was the only symptom suggestive of the emetic activity seen in swine. The birds then developed diarrhea and made no

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[2] The use of trade names in this publication does not imply endorsement by the North Carolina Agricultural Research Service nor criticism of similar ones not mentioned.

TABLE 1. *Mortality caused by vomitoxin in young broiler chickens*

Vomitoxin (mg/kg)	Mortality[a]	Survival time (hr)
0	0/2	. . .
35	0/2	. . .
70	0/2	. . .
140	1/2	13.5
280	2/2	13.5
560	2/2	3.5
1120	2/2	3.7

[a]Mortality is expressed as deaths per number of birds injected.

attempt to approach or use the feeder and waterer. About 30 min prior to death the birds became prostrate, and intermittent, tonic convulsions ensued. Immediately preceding death, breathing became erratic with episodes of hyperventilation, giving the impression of death through respiratory failure. Deaths occurred between 3.5 and 13.5 hr after injection.

Necropsy of the cadavers revealed that the most prominent lesion was ecchymotic hemorrhaging throughout the intestinal tract (Fig. 1), liver (Fig. 2), and musculature (Fig. 3). The hemorrhaging was so extensive and intensive that the carcass could be described as burgundy colored. Although the kidneys appeared normal, visceral gout with its attendant deposits of urates was present suggesting kidney dysfunction. Urate deposition was so extensive that it even occurred subcutaneously (Fig. 3). The crops were distended with gas and some necrotic lesions of the gizzard lining, suggestive

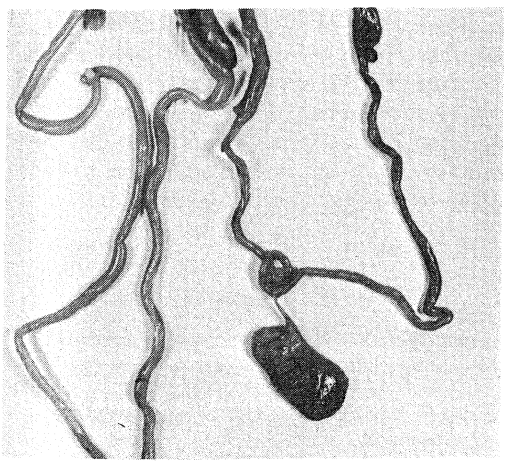

FIG. 1. Ecchymotic hemorrhaging of the intestinal tract caused by vomitoxin. Intestines from a control bird are on the left and from a treated bird are on the right.

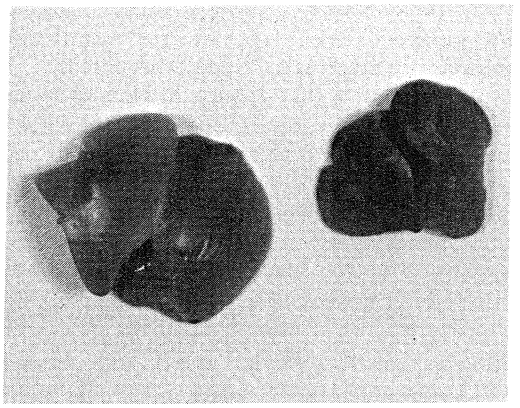

FIG. 2. Ecchymotic hemorrhaging of the liver caused by vomitoxin. The liver on the left is from a control bird and the liver on the right is from a treated bird.

FIG. 3. Ecchymotic hemorrhaging in the musculature of a bird treated with vomitoxin. A whitish speckled area of extensive urate deposition is on the abdomen posterior to the keel bone.

of an inflammatory response, were apparent. The survivors after 7 days appeared normal and on necropsy no gross pathology was noted.

It is intriguing that vomitoxin elicited frank hemorrhaging and urate deposition consistent with the description by Forgacs and Carll (1962) of the historically and economically important hemorrhagic anemia syndrome of chickens caused by moldy feed. Vomitoxin appears to be the first mycotoxin reported to cause in chickens the frank hemorrhaging characteristic of hemorrhagic anemia syndrome despite extensive evaluations (Doerr *et al.*, 1974). An approximate LD_{50} of 140 mg/kg for vomitoxin suggests substantially less toxicity than aflatoxin and ochratoxin with their LD_{50} values of 6.8 and 2.1 mg/kg, respectively (Smith and Hamilton, 1970; Huff *et al.*, 1974). Nevertheless, investigations into the possible relationship of vomitoxin to hemorrhagic anemia syndrome and into any interactions vomitoxin might have with other mycotoxins seem warranted.

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